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#32	Link to **PubMed** from (**7593158**)	11:14:04	0
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#13	Search **3.4.24 AND human AND disintegrin**Field: **Title/Abstract Word**	10:18:40	0
#12	Search **3.4.24 AND human AND disintegrin**	10:18:20	131
#11	Search **3.4.24 AND human AND pituitary**	10:16:56	18
#10	Search **3.4.24 AND human**	10:16:48	7422
#9	Search **3.4.24**	10:16:39	13661
#8	Search **ADAM AND human AND protease AND disintegrin**	09:23:44	62
#7	Search **ADAM AND human AND protease**	09:23:11	153

#6 Search **ADAM AND human**	09:23:00	3158
#5 Search **ADAM and human**	09:22:54	3158
#4 Search **3.4.24.46[EC/RN Number]**	09:22:28	17
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(FILE 'HOME' ENTERED AT 09:24:47 ON 01 JUN 2001)

FILE 'CANCERLIT, MEDLINE, CAPLUS, BIOSIS' ENTERED AT 09:25:06 ON 01 JUN 2001

L1	0	S	ADAM AND WOLFSBERG
L2	283	S	ADAM AND DISINTEGRIN AND METALLOPROTEASE
L3	173	S	ADAM (3N) DISINTEGRIN (3N) METALLOPROTEASE
L4	80		DUPLICATE REMOVE L3 (93 DUPLICATES REMOVED)
L5	0	S	L4 (5N) CELL-MATRIX

FILE 'MEDLINE' ENTERED AT 09:27:55 ON 01 JUN 2001

		E	WOLFSBERG T/AU 25
L6	0	S	(E3) AND (ADAM)
		E	MYLES D/AU 25
L7	10	S	(E7) AND (ADAM)

FILE 'STNGUIDE' ENTERED AT 09:30:53 ON 01 JUN 2001

FILE 'MEDLINE' ENTERED AT 09:31:13 ON 01 JUN 2001

L8	0	S	DISTINTEGRIN (5N) METALLOPROTEASE

FILE 'CANCERLIT, MEDLINE, CAPLUS, BIOSIS' ENTERED AT 10:24:55 ON 01 JUN 2001

L9	0	S	DISTINTEGRIN (5N) METALLOPROTEASE
L10	0	S	DISTINTEGRIN AND METALLOPROTEASE
L11	0	S	DISTINTEGRIN AND PROTEASE
L12	6	S	DISTINTEGRIN
L13	8830	S	ADAM
L14	143	S	L13 AND PROTEASE
L15	68		DUPLICATE REMOVE L14 (75 DUPLICATES REMOVED)
L16	34	S	L15 AND HUMAN
L17	1206	S	MDC
L18	9	S	L17 AND PROTEASE
L19	3		DUPLICATE REMOVE L18 (6 DUPLICATES REMOVED)

L28 ANSWER 6 OF 18 CANCERLIT
AN 2000004301 CANCERLIT
DN 20004301
TI Expression of Bacteroides fragilis virulence markers in vitro.
AU Ferreira R; Alexandre M C; Antunes E N; Pinhao A T; Moraes S R; Ferreira M
C; Domingues R M
CS Instituto de Microbiologia, UFRJ, Rio de Janeiro, Brazil.
SO JOURNAL OF MEDICAL MICROBIOLOGY, (1999). Vol. 48, No. 11, pp. 999-1004.
Journal code: J2N. ISSN: 0022-2615.
DT Journal; Article; (JOURNAL ARTICLE)
FS MEDL; L; Priority Journals
LA English
OS MEDLINE 20004301
EM 199912
AB Bacteroides fragilis isolates from intestinal and non-intestinal infections, normal flora and the environment were examined for properties linked with interactions among cells in vitro. Different **adhesion** molecules were detected in agglutination assays with **human** erythrocytes and tests for auto-agglutination and adherence to **human** colon carcinoma cells (HT29). There was no correlation between these properties, indicating that independent molecules are involved. Treatment with trypsin, heat or EDTA inhibited agglutination and
adherence, suggesting that these molecules are proteins. The lack of correlation with the origin of the strains did not permit any of these activities to be recognised as virulence markers. The expression of fragilysin, a **protease** associated with damage to intestinal cells and bacterial translocation, was examined. Only those strains from patients with diarrhoea expressed this **protease** activity in assays with HT29 cells and this was confirmed by specific PCR for the bft gene. The activity of fragilysin as an enterotoxin was confirmed in the rabbit intestinal ligated loop assay. The association of this property only with strains from intestinal infections indicates that it is too early to suggest this **protease** as a determinant factor of B. fragilis invasiveness.

L28 ANSWER 7 OF 18 CANCERLIT
AN 1999146296 CANCERLIT
DN 99146296
TI Actions of heparin that may affect the malignant process.
AU Engelberg H
CS California Arteriosclerosis Research Foundation, Beverly Hills 90210, USA.
SO CANCER, (1999). Vol. 85, No. 2, pp. 257-72.
Journal code: CLZ. ISSN: 0008-543X.
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)
FS MEDL; L; Abridged Index Medicus Journals; Priority Journals; Cancer Journals
LA English
OS MEDLINE 99146296
EM 199903
AB BACKGROUND: Heparin has many actions that may affect the malignant process, especially metastasis. METHODS: The author conducted an extensive review of the available medical literature about heparin activity that may apply to important factors involved in the malignant process. RESULTS: Thrombin is generated by tumors, and the resultant fibrin formation impedes natural killer cell activity. Microthrombi arrest tumor cells in capillaries. Heparin prevents the formation of thrombin and neutralizes its activity. Angiogenesis has an important role in metastasis; heparin minimizes angiogenesis via the inhibition of vascular endothelial growth factor, tissue factor, and platelet activating factor. It decreases tumor cell **adhesion** to vascular endothelium as it inhibits selectin and chemokine actions, and it also decreases the replication and activity of some oncogenic viruses. Matrix metalloproteinases, serine **proteases**, and heparanases have an important role in metastasis. Heparin decreases their activation and limits their effects. It competitively inhibits tumor cell attachment to heparan sulfate proteoglycans. It blocks the oncogenic action of ornithine decarboxylase and enhances the antineoplastic effect of transforming growth factor-beta.
Heparin inhibits activator protein-1, which is the nuclear target of many oncogenic signal transduction pathways, and it potently inhibits casein kinase II, which has carcinogenic activity. Platelet-derived growth factor, which has oncogenic effects, is also inhibited by heparin, as are reverse transcriptase, telomerase, and topoisomerase prooncogenic actions.
CONCLUSIONS: These various heparin actions justify clinical investigation of its possible beneficial effect on malignant disease.

L28 ANSWER 8 OF 18 MEDLINE
AN 1999218159 MEDLINE
DN 99218159 PubMed ID: 10189392
TI Inflammation, sepsis, and coagulation.
AU Esmon C T; Fukudome K; Mather T; Bode W; Regan L M; Stearns-Kurosawa D J; Kurosawa S
CS Oklahoma Medical Research Foundation Cardiovascular Biology Research, 825 N.E. 13th Street, Oklahoma City, Oklahoma 73104, USA..
esmonc@omrf.ouhsc.edu
NC PO1 HL54804 (NHLBI)
R37 HL 30340 (NHLBI)
SO HAEMATOLOGICA, (1999 Mar) 84 (3) 254-9. Ref: 24

Journal code: FYB; 0417435. ISSN: 0390-6078.
CY Italy
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 199906
ED Entered STN: 19990712
Last Updated on STN: 19990712
Entered Medline: 19990623
AB The molecular links between inflammation and coagulation are unquestioned.
Inflammation promotes coagulation by leading to intravascular tissue factor expression, eliciting the expression of leukocyte **adhesion** molecules on the intravascular cell surfaces, and down regulating the fibrinolytic and protein C **anticoagulant** pathways. Thrombin, in turn, can promote inflammatory responses. This creates a cycle that logically progresses to vascular injury as occurs in septic shock. Most complex systems are regulated by product inhibition. This inflammation-coagulation cycle seems to follow this same principle with the protein C pathway serving as the regulatory mechanism. The molecular basis by which the protein C pathway functions as an **anticoagulant** is relatively well established compared to the mechanisms involved in regulating inflammation. As one approach to identifying the mechanisms involved in regulating inflammation, we set out to identify novel receptors that could modulate the specificity of APC in a manner analogous to the mechanisms by which thrombomodulin modulates thrombin specificity. This approach led to the identification of an endothelial cell protein C receptor (EPCR). To understand the mechanism, we obtained a crystal structure of APC (lacking the Gla domain). The crystal structure reveals a deep groove in a location analogous to anion binding exosite 1 of thrombin, the location of interaction for thrombomodulin, platelet thrombin receptor and fibrinogen. Thrombomodulin blocks the activation of platelets and fibrinogen without blocking reactivity with chromogenic substrates or inhibitors. Similarly, in solution, EPCR blocks factor Va inactivation without modulating reactivity with **protease** inhibitors. Thus, these endothelial cell receptors for the protein C system share many properties in common including the ability to be modulated by inflammatory cytokines. Current studies seek to identify the substrate for the APC-EPCR complex as the next step in elucidating the mechanisms by which the protein C pathway modulates the response to injury and inflammation.

L28 ANSWER 12 OF 18 CANCERLIT
AN 1998281176 CANCERLIT
DN 98281176
TI A subcloned **human** esophageal squamous cell carcinoma cell line with low thrombomodulin expression showed increased invasiveness compared with a high thrombomodulin-expressing clone--thrombomodulin as a possible candidate for an **adhesion** molecule of squamous cell carcinoma.
AU Matsushita Y; Yoshiie K; Imamura Y; Ogawa H; Imamura H; Takao S; Yonezawa S; Aikou T; Maruyama I; Sato E
CS Department of Pathology II, Faculty of Medicine, Kagoshima University, Japan.
SO CANCER LETTERS, (1998). Vol. 127, No. 1-2, pp. 195-201.
Journal code: CMX. ISSN: 0304-3835.
DT Journal; Article; (JOURNAL ARTICLE)
FS MEDL; L; Priority Journals; Cancer Journals
LA English
OS MEDLINE 98281176
EM 199807
AB Thrombomodulin (TM) is an endothelial cell surface glycoprotein which converts thrombin from a procoagulant **protease** to an **anticoagulant**. We have previously reported that TM is a useful marker for immunohistochemical diagnosis of angiogenic tumors and also have reported that TM is expressed on squamous cell carcinoma (SCC) of the **human** esophagus. In addition, the expression of TM is significantly decreased in metastatic foci in lymph nodes compared with that in primary lesions. In order to reveal the biological significance of TM in SCC, we subcloned and established two different cell lines, i.e. TM-high-expressing (TE3HTM) cells and TM-low-expressing (TE3LTM) cells, from a **human** SCC cell line, TE3, using fluorescence-activated cell sorter (FACS) and examined the biological characteristics of these variant cell lines. These tumor cells revealed very similar morphological figures in ordinary cultured conditions and showed almost equal growth rates under various cultured conditions. By the invasion assay of these tumor cells using matrigel, we found that TE3LTM cells showed significantly increased invasive ability compared with that of TE3HTM cells. Characteristic intercellular localization of TM and a different manner of invasiveness between TE3LTM cells and TE3HTM cells suggest that TM may act as a cell-to-cell interaction molecule.

L28 ANSWER 15 OF 18 MEDLINE
AN 95078397 MEDLINE
DN 95078397 PubMed ID: 7986950
TI Immobilization of **human** thrombomodulin onto poly(ether urethane urea) for developing antithrombogenic blood-contacting materials.
AU Kishida A; Ueno Y; Fukudome N; Yashima E; Maruyama I; Akashi M
CS Department of Applied Chemistry and Chemical Engineering, Faculty of Engineering, Kagoshima University, Japan.
SO BIOMATERIALS, (1994 Aug) 15 (10) 848-52.
Journal code: A4P; 8100316. ISSN: 0142-9612.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199501
ED Entered STN: 19950124
Last Updated on STN: 19960129
Entered Medline: 19950106
AB Thrombomodulin (TM) is a newly described endothelial cell-associated protein that functions as a potent natural **anticoagulant** by converting thrombin from a procoagulant **protease** to an **anticoagulant**. In this study, focussing on the application of TM for biomedical materials, recombinant **human** TM (hTM) was immobilized onto the polymers for medical use, and the evaluation of their antithrombogenicity and the interaction with platelets were investigated. As the base polymer for immobilization reaction, poly(ether urethane urea) (PEUU), which was reported to have good blood compatibility, was used. hTM-immobilized PEUU showed superior antithrombogenic activity, such as the prolongation of plasma recalcification time and the inhibition of thrombin-induced platelet aggregation, though the amount of immobilized hTM was very small (i.e. less than 1 microgram/cm2). Platelet **adhesions** onto hTM-immobilized PEUU were not observed. These results show that the immobilization of hTM does not change the native good blood compatibility of PEUU, but provides excellent **anticoagulant** activity.

L28 ANSWER 16 OF 18 MEDLINE
AN 94331839 MEDLINE
DN 94331839 PubMed ID: 7519910
TI **Human** protein C inhibits selectin-mediated cell **adhesion** : role of unique fucosylated oligosaccharide.
AU Grinnell B W; Hermann R B; Yan S B
CS Cardiovascular Research, Lilly Research Laboratories, Eli Lilly and Company, Indianapolis, IN 46285-1543.
SO GLYCOBIOLOGY, (1994 Apr) 4 (2) 221-5.
Journal code: BEL; 9104124. ISSN: 0959-6658.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199409
ED Entered STN: 19940920
Last Updated on STN: 19960129
Entered Medline: 19940914
AB The **human anticoagulant** factor, Protein C, is a plasma glycoprotein that has reported anti-ischaemic and anti-inflammatory properties. To explore potential mechanisms for these reported activities,

we examined the effect of Protein C on the process of cell **adhesion** to vascular endothelial cells, which plays a critical role during inflammatory responses. We show that both **human** plasma-derived and **human** cell-produced recombinant Protein C inhibit E-selectin-mediated cell **adhesion**. This effect was not mediated through the serine **protease** activity of Protein C, but through its carbohydrates. Using oligosaccharides isolated from **human** cell-produced Protein C, we have defined a polylactosamine structural determinant that inhibits **adhesion**. This uncharged determinant appears to be a more potent ligand for E-selectin than the sialylated Lewis X antigen. Our data suggest a potential mechanism for the reported anti-inflammatory effects of Protein C and describe a new ligand for selectin-mediated **adhesion**.

L28 ANSWER 17 OF 18 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1994:205350 BIOSIS
DN PREV199497218350
TI Interactions between blood and vascular wall: Antithrombotic versus prothrombotic mechanisms.
AU Gerlach, E. (1); Becker, B. F.
CS (1) Physiologisches Institut der Universitet Muenchen, Pettenkoferstr. 12,
D-80336 Muenchen Germany
SO Zeitschrift fuer Kardiologie, (1993) Vol. 82, No. SUPPL. 5, pp. 13-21. ISSN: 0300-5860.
DT General Review
LA German
SL German; English
AB The coagulation enzyme thrombin, a serine **protease** like all other coagulation factors, plays a central role in the hemostatic processes engaged after injurious events. It induces, with particular efficacy, the aggregation of blood platelets (primary hemostasis) and accounts, via splitting of fibrinogen to fibrin, for the event actually responsible for the coagulation of blood (secondary hemostasis). As is well-known, thrombin itself is generated by a cascade of activation events involving various coagulation factors (F). In this respect the "tissue factor" (TF, formerly known as thromboplastin), in combination with FVIIa, attains decisive significance, not only in the extrinsic pathway of coagulation (activation of F X fwdarw Xa), but also in the intrinsic pathway (activation of F IX fwdarw IXa). Under physiological circumstances, platelet aggregation and coagulation are restricted to the area of the vascular lesion, since the surrounding intact endothelium inhibits an intraluminal spreading of both processes. These "antithrombotic" features of the endothelium encompass antiaggregatory mechanisms (formation and release of prostacyclin (PGI-2), adenosine, EDRF (NO), degradation of ADP and other nucleotides mediated by ecto-nucleotidases) as well as anti-coagulatory properties (formation and release of "tissue factor pathway inhibitor" (TFPI), which blocks the coagulation cascade by joining F Xa, TF and F VIIa into an inactive complex, thrombomodulin - thrombin induced activation of protein C, which, together with protein S, inactivates F Va and F VIIIa, thereby attenuating further generation of thrombin, and the heparan sulfate-enhanced activation of antithrombin III and heparin-cofactor II). Arteriosclerotic and inflammatory alterations of the vessel wall lead to endothelial dysfunction in the perturbed sections. This is expressed both as a weakening of the anti-thrombogenic features and the development of prothrombotic characteristics. Particular emphasis must be placed on TF, expressed on the endothelial cell surface and/or released into the subendothelial matrix, thereby initiating a local formation of thrombin at the site of the mural perturbation. Recent studies suggest that it is this very thrombin which contributes to the progression of the arteriosclerotic lesion, by way of multiple effects on the endothelium (enhanced expression of TF, expression and externalization of leukocyte **adhesion** molecules, increased vascular permeability, formation and release of

PGI-2, NO, PAF, PDGF, endothelin, PAI-1, TPA, vWF, etc.). In atherosclerotic plaques, the monocyte derived macrophages (foam cells) are richly endowed with TF. Consequently, plasma insudation resulting from the disturbed endothelial barrier function can lead to accelerated formation of thrombin and consecutive fibrin deposition within the plaque. Development of fissures or ruptures of plaques causes, on account of the TF-dependent activation of the coagulation process on the surface of macrophages, a focal, massive production of thrombin. This leads, in turn, to the acute formation of a platelet- and fibrin-containing clot, instigating partial or total vascular occlusion. - Modern therapeutic concepts are aimed at directly counteracting the multifunctional actions of thrombin; heparin, hirudin, hirulog, arginine-containing tripeptides such as argatroban, and thrombomodulin are proven agents or promising candidates.

L32 ANSWER 5 OF 6 MEDLINE
AN 88294142 MEDLINE
DN 88294142 PubMed ID: 2840973
TI Pro-opiomelanocortin and pro-vasopressin converting enzyme in **pituitary** secretory vesicles.
AU Loh Y P; Birch N P; Castro M G
CS Laboratory of Neurochemistry and Neuroimmunology, National Institute of Child Health and Human Development, Bethesda, MD 20892.
SO BIOCHIMIE, (1988 Jan) 70 (1) 11-6.
Journal code: A14; 1264604. ISSN: 0300-9084.
CY France
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198809
ED Entered STN: 19900308
Last Updated on STN: 20000303
Entered Medline: 19880912
AB Peptide hormones are synthesized from larger precursors by cleavages at paired basic residues. We have isolated a pro-hormone converting enzyme from bovine neural and intermediate lobe secretory vesicles that cleaves pro-vasopressin and pro-opiomelanocortin at Lys-Arg residues to yield vasopressin, and adrenocorticotropin/endorphin-related peptides, respectively. The enzyme from both lobes is an aspartyl protease of approximately 70,000 Da, is a glycoprotein and has an optimum pH range of 4.0-5.0. Present within the same secretory vesicles is an aminopeptidase B-like enzyme which is a **metalloprotease** that is inhibited by Co2+ and Zn2+. This enzyme may play a role in trimming off the N-terminal extended basic residues from peptides liberated by the pro-hormone converting enzyme.

L16 ANSWER 23 OF 34 MEDLINE
AN 1999192284 MEDLINE
DN 99192284 PubMed ID: 10094461
TI ADAMTS: a novel family of **proteases** with an **ADAM** **protease** domain and thrombospondin 1 repeats.
AU Tang B L; Hong W
CS Membrane Biology Laboratory, Institute of Molecular and Cell Biology, Singapore, Singapore.. mcbtbl@imcb.nus.edu.sg
SO FEBS LETTERS, (1999 Feb 26) 445 (2-3) 223-5. Ref: 15
Journal code: EUH; 0155157. ISSN: 0014-5793.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 199904
ED Entered STN: 19990504
Last Updated on STN: 20000303
Entered Medline: 19990420